CLAIMS

1. A method for delivering a therapeutic dose of a gene expression cassette in a fluid selectively to heart for sustained expression comprising steps of:

(a) increasing dwell time of fluid in a targeted area,

(b) administration of a vascular permeablizing agent, and

(c) administration of a viral vector containing a gene expression cassette of interest.

2. A method as in claim 1, wherein the dwell time is increased by the induction of hypothermia.

3. A method as in claim 1, wherein the dwell time is increased by isolation of the heart from systemic circulation.

4. A method as in claim 1, wherein the dwell time is increased by induction of hypothermia and isolation of the heart from systemic circulation.

5. A method as in claim 1, wherein dwell time is increased by induction of complete or near-complete transient cardiac arrest.

6. A method as in claim 1, wherein dwell time is increased by induction of reversible bradycardia.

7. A method as in claim 1, wherein the vascular permeablizing agent is histamine, substance P or serotonin.

8. A method as in claim 1, wherein at least one bolus of virus is administered.

9. A method as in claim 1, wherein the viral vector is an adenoviral vector.

10. A method as in claim 9, wherein the adenoviral vector contains a strong promoter.

11. A method as in claim 10, wherein the strong promoter is a cytomegalovirus (CMV) promoter.

12. A method as in claim 10, wherein the strong promoter is a Rous sarcoma virus (RSV) promoter.

13. A method as in claim 9, wherein the adenoviral vector contains enhancer elements.

14. A method as in claim 13, wherein the enhancer is a cytomegalovirus (CMV) enhancer.

15. A method as in claim 13, wherein the enhancer is a Rous sarcoma virus (RSV) enhancer.

16. A method as in claim 1, wherein the viral vector is an adenovirus-associated viral (AAV) vector.

17. A method as in claim 16, wherein the AAV vector contains a strong promoter.

18. A method as in claim 17, wherein the strong promoter is a cytomegalovirus (CMV) promoter.

19. A method as in claim 16, wherein the strong promoter is a Rous sarcoma virus (RSV) promoter.

20. A method as in claim 9, wherein the AAV vector contains enhancer elements.

21. A method as in claim 20, wherein the enhancer is a cytomegalovirus (CMV) enhancer.

22. A method as in claim 20, wherein the enhancer is a Rous sarcoma virus (RSV) enhancer.

23. A method as in claim 1, wherein the gene of interest is a structural gene.

24. A method as in claim 23, wherein the structural gene is α-sarcogylcan.

25. A method as in claim 23, wherein the structural gene is β-sarcogylcan.

26. A method as in claim 23, wherein the structural gene is γ-sarcogylcan.

27. A method as in claim 23, wherein the structural gene is δ-sarcogylcan.

28. A method as in claim 1, wherein the gene of interest is a functional gene.

29. A method as in claim 28, wherein the functional gene is β-adrenergic receptor (β-AR).

30. A method as in claim 28, wherein the functional gene is sarcoplasmic reticulum Ca^{2+} ATPase (SERCA-2).

31. A method as in claim 1, wherein the gene of interest is a gene fragment.

32. A method as in claim 1, wherein the gene of interest is a mutated form of a gene.

33. A method as in claim 32, wherein the mutated form of the gene is a dominant negative form of phospholamban (PLB).

34. A method as in claim 32, wherein the SERCA-2 gene is administered in conjunction with a dominant negative form of PLB.

35. A method as in claim 33, wherein the dominant negative form of PLB contains a mutation at amino acid 2 from glutamic acid (E) to alanine (A).

36. A method as in claim 33, wherein the dominant negative form of PLB contains a mutation at amino acid 14 from arginine (R) to glutamic acid (E).

37. A method as in claim 33, wherein the dominant negative form of PLB contains a mutation at amino acid 16 from serine (S) to asparagine (N).

38. A method as in claim 33, wherein the dominant negative form of PLB contains mutations at amino acid 16 from serine (S) to glutamic acid (E).

39. A method as in claim 33, wherein the dominant negative form of PLB contains a mutation at amino acid 49 from valine (V) to alanine (A).

40. A method as in claim 33, wherein the dominant negative form of PLB contains mutations at amino acid 3 from lysine (K) to glutamic acid (E) and at amino acid 14 from arginine (R) to glutamic acid (E).